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Related Articles, Links

An N-terminal peptide from p60src can direct myristylation and plasma membrane localization when fused to heterologous proteins.

Pellman D, Garber EA, Cross FR, Hanafusa H.

The src gene product, p60src, of Rous sarcoma virus (RSV) is a tyrosine-specific protein kinase which is associated with the plasma membrane of infected cells. Myristic acid is bound in an amide linkage to glycine 2 of p60src. Of the N-terminal 30 kilodaltons of p60src, only amino acids 1-14 are required for myristylation, and myristylation of p60src may be required for its membrane association, and for cell transformation. To test the hypothesis that the first 14 amino acids of p60src contain a recognition sequence for myristylation, we have fused the DNA sequence coding for these amino acids to either the fps gene of the F36 derivative of Fujinami sarcoma virus (FSV), or to the chimpanzee alpha-globin gene. We report here that although the fusion proteins were myristylated, the parental proteins were not, and unlike the non-myristylated F36 p91fps which was not bound to the plasma membrane, the myristylated fusion protein was bound, like p60src. We conclude that the first 14 amino acids of p60src contain a sequence which is sufficient for myristylation, and which may direct proteins to the plasma membrane.

PMID: 3920530 [PubMed - indexed for MEDLINE]

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1: J Biol Chem 1994 Jul 29;269(30):19203-6

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The CAAX peptidomimetic compound B581 specifically blocks farnesylated, but not geranylgeranylated or myristylated, oncogenic ras signaling and transformation.

Cox AD, Garcia AM, Westwick JK, Kowalczyk JJ, Lewis MD, Brenner DA, Der CJ.

Department of Pharmacology, Lineberger Comprehensive Cancer Center, University of North Carolina, Chapel Hill 27599.

Recently developed CAAX peptidomimetic compounds have been shown to be potent and specific inhibitors of farnesyl protein transferase activity and to block the growth of Ras-transformed cells. However, whether this growth inhibitory action is specifically a consequence of blocking oncogenic Ras signaling has not been determined. To address this question, we have utilized mutants of the normally farnesylated oncogenic Ras protein (Ras-F) that are modified by alternative lipids, a geranylgeranyl isoprenoid (Ras-GG) or the fatty acid myristate (Myr-Ras), to determine the specificity of the CAAX peptidomimetic compound, B581. Like Ras-F, both Ras-GG and Myr-Ras are membrane-associated and transforming. Unexpectedly, NIH 3T3 cells transformed by each of the three Ras mutants underwent morphological alteration to a less transformed, but not normal, morphology. However, B581 inhibited the ability of only Ras-F-transformed cells, but not Ras-GG- or Myr-Ras- (or Raf-) transformed cells, to grow in soft agar. Furthermore, although all three lipid-modified versions of Ras stimulated mitogen-activated protein kinase activation, and both Jun and Elk-1 transcriptional activity, B581 inhibited only farnesylated Ras activation of these three downstream components of Ras signaling. Therefore, B581 prevents the growth of Ras-transformed cells by specifically antagonizing Ras-mediated signaling.

PMID: 8034681 [PubMed - indexed for MEDLINE]

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